

Charcot the malady is not an entity, but a symptomatic complex, characterized by the presence of hemicrania with concurrent total paralysis of one of the oculomotor nerves. The one sided head-ache is quite characteristic, and emphasized by the various observers. The intensity of the pain is seldom very great, but deeply seated, continuous, with morning or evening exacerbations, and extending from the occiput to the orbit. The ocular paralysis sets in after the development of hemicrania; it is invariably unilateral, and nearly always a complete palsy. Another characteristic feature, observed by all, is the tendency and periodicity of relapses, as for instance, once or twice yearly, every three months, etc. In one case (Moebius) every August for three consecutive years, in another, (Thomsen) in May and October. After a certain length of time, the disease undergoes a change, the symptoms becoming more permanent and continuous. 1. Periodical stage; 2 Permanent stage. A typical attack of *migraine ophthalmoplegique* presents, during the *painful period*, the same features as ordinary migraine (nausea, vomiting, photophobia and hemicrania), at times with oedema of the eyelids, and salivation. The duration is quite variable and sooner or later exacerbations intervene. The onset is generally sudden without any definite cause.

The *paralytic period* sets in generally several hours or days, or, rarely, weeks after the painful and gastric symptoms. The upper eyelid begins to droop, the bulb deviates externally and the whole distribution of one of the third nerves is impaired (paralysis of the iris, the muscles of accommodation as well as the external eye muscles). The development of these phenomena is rapid without being sudden as in cerebral hemorrhage. The painful symptoms (migraine) usually continue during the paralytic stage for some time, but are less marked or intense, which fact is in opposition to Charcot, who considered the onset of the latter period as indicative of cessations of the former. The paralytic troubles may last for hours or months, disappearing as they set in, rapidly, the patient feeling as well as ever after the attack. New attacks are always to be anticipated, which invariably have the same characteristics as the preceding one.

As the malady grows older it takes a chronic character (Thomsen's case lasted for 29 years), the paralytic attacks becoming rarer but more tenacious, and in the intervals between, the symptoms persistent with periods of exacerbations, which gives the malady the character of a continuous disorder with periodical exacerbations. The tendency to become aggravated is far greater than to recover, although there may be a possibility of a spontaneous cure. The ætiology and pathology are quite obscure, although the subject of considerable speculation. The treatment consists in the prolonged administration of bromides (Charcot) with potassii iodide for circulatory disturbances, and electricity. The last especially when the paralysis of the eye muscles is permanent.

MACALESTER.

A CASE OF TABES DORSALIS WITH BULBAR SYMPTOMS.

Deutsche Zeitschrift für Nervenheilkunde. Vol. 9, Nos. 1 and 2, 1896. By H. Grabower, M. D.

In a case of tabes the following conditions were found. Degeneration of the left recurrent nerve and of the extrabulbar portion of the roots of both tenth nerves, perfect integrity of the roots of both eleventh nerves, degeneration of the roots of both ninth nerves, integrity of the nuclei and intrabulbar portion of the roots of the tenth and eleventh nerves, moderate degeneration of both spinal roots of the fifth nerves. The nuclei of the third, fourth and sixth nerves were atrophied.

The author is inclined to accept the theory that in certain cases the tabetic process begins in the peripheral sensory nerves, and ascends to

the posterior roots and posterior columns. The spinal ganglia may be altered functionally without presenting organic changes.

On account of the disease of the sensory nerves, the cells of the spinal ganglia are deprived of the normal impulses from the periphery, and undergo the atrophy of inactivity, which however may not be demonstrable (Marinesco).

To explain the involvement of the motor nerves, as of the vagus in this case when its nucleus was intact, it is necessary to suppose that the morbid process is transmitted by means of the sensory roots to the cells of the anterior horns in the cord, and within the oblongata to the motor nuclei. These are altered functionally, but not organically. In this case the sensory descending roots of the ninth and fifth nerves were degenerated.

The case is of great value in regard to the motor innervation of the larynx. It has been a disputed question as to whether this is a function of the vagus or of the accessorius. In 1890 Grabower from experiments reached the conclusion that the accessorius has absolutely nothing to do with the innervation of the larynx, and that the vagus is its only motor nerve. Similar statements were made by Gros-man.

Grabower has shown by means of serial sections that the nucleus of the accessorius passes into that of the hypoglossus, that a cerebral accessorius nucleus does not exist, that from where the nucleus of the accessorius terminates to where the motor nucleus of the vagus begins there is a space of more than nine millimetres, and that there is not the slightest connection between the vagus and accessorius nuclei.

There are sixteen cases known to Grabower in which paralysis of the larynx was observed in connection with paralysis of the trapezius. Seven of these are undoubtedly of peripheral origin and useless therefore for the question at issue, for a process which involves the accessorius after its exit from the jugular foramen is apt to involve the vagus also. In none of the nine remaining cases has an autopsy been reported.

In Grabower's case of paralysis of the laryngeal muscles the extrabulbar part on of the roots of the tenth nerves were found almost completely atrophied, while the roots of the eleventh nerves were perfectly intact. This according to the author, proves that the accessorius has nothing to do with the innervation of the larynx.

Oppenheim in speaking of this case, says that Grabower has demonstrated that the innervation of the larynx is through the vagus and not through the accessorius. He believes the tabetic virus affected the vagi roots, and not the nuclei in a manner similar to the action of lead on the radial nerve, although he acknowledges the difficulty in deciding this point.

Pharyngeal crises, which were observed in this case, were probably due to the degeneration of the ninth nerves.

The degeneration of the spinal roots of the fifth nerves explains the analgesia of the face and the loss of the teeth observed in this case.

SPILLER.

ARTHROPATHY AS THE EARLIEST SYMPTOM OF TABES DORSALIS.

F. DE GRANDMAISON (*Médecine Moderne*, Sept. 30, 1896,) describes two cases in which the earliest conspicuous symptoms of ataxia were the appearance of articular enlargements at the knee. One patient was a man of fifty years, the other a woman of thirty-five. The diagnosis of ataxia was made in both cases on two cardinal symptoms, the total absence of knee-jerk and the presence of the Argyll Robertson phenomenon. Neither patient had any disorder of station, neither had suffered with any crises of pain, so that both may be considered to have had remarkably early articular manifestations, although it will be remembered that Charcot insisted upon the frequency with which arthropathies appeared in the pre-ataxic stage.

MITCHELL.